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THE AUTUMN MEETING of the British Cardiac Society was held at the Wembley Conference Centre on Thursday and Friday, 1 and 2 November 1979. The President, W SOMERVILLE, took the Chair during private business. At the scientific sessions the Chair was taken by LAWSON MCDONALD.

Abstracts of papers

YOUNG RESEARCH WORKERS AWARD

Tissue acidosis in myocardial ischaemia

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The significance of acidosis in ischaemic heart muscle was investigated in isolated rabbit myocardium, in open-chested dogs, and in patients with coronary artery disease. A needle and catheter tip electrode were designed and developed for continuous recording of myocardial tissue pH in animals, and of intravascular pH in the canine and human coronary sinus.

In isolated rabbit myocardium tissue pH fell within five seconds of the onset of ischaemia; the fall after 30 minutes ischaemia was more than 1 pH unit. In dogs, during 150 seconds of coronary occlusion a rapid fall in tissue pH also occurred, but coronary venous pH was almost unchanged. On reperfusion, however, there was a large transient fall in coronary venous pH caused by washout of hydrogen ions from the tissue.

Coronary sinus and arterial pH were continuously monitored during and after atrial pacing in 20 patients. In nine patients with evidence of ischaemia on pacing, coronary sinus pH fell during pacing ($P < 0{\cdot}001$), but the greatest fall was in the 30 seconds after the end of pacing, analogous to the washout seen in dog experiments. Coronary sinus pH did not fall in patients without ischaemia ($\chi^2 = 11{\cdot}4$, $P < 0{\cdot}001$).

Tissue acidosis can account for most of the early fall in contractility during myocardial ischaemia. Continuous measurement of coronary sinus pH during and immediately after atrial pacing gives direct evidence of ischaemia in man.

Improvement of electrocardiogram by chronic beta adrenoceptor blockade during healing phase of clinical and experimental myocardial infarction

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I have developed a new technique for praecordial electrocardiographic mapping to quantify ischaemic myocardial damage and validated it by showing a correlation ($r = 0{\cdot}827$ and $r = -0{\cdot}623$) between the maximum ΣQ and minimum ΣR from the map compared with the cumulative release of CK MB in 28 patients with anterior myocardial infarction. The reproducibility of the technique was studied under a wide variety of conditions.

Mapping was then used to study the evolution of the electrocardiogram in the chronic healing phase in 22 patients with acute myocardial infarction randomised to receive atenolol or placebo for 12 months. A significantly greater recovery of ΣR wave and disappearance of ΣQ was observed in the treated group compared with those on placebo ($2p < 0{\cdot}001$ and $2p < 0{\cdot}025$, respectively).

A further group of 67 patients with acute myocardial infarction randomised between metoprolol and placebo were studied using standard 12 lead electrocardiograms. This showed that the return to normal of the electrocardiogram was related to slow heart rates either spontaneous or induced by beta-blockers. An initial small infarct was found to be associated with improved recovery on the electrocardiogram.

I have also shown improved recovery on electrocardiogram with atenolol in 14 dogs after acute myocardial infarction.

I have evidence that these changes are not the result of intraventricular conduction defects, bradycardia *per se*, left ventricular hypertrophy, or cardiac dilatation. It may be a result of improved healing or shrinkage of myocardial scar tissue.

Relation of ventricular arrhythmias to primary ventricular fibrillation

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Potential 'warning arrhythmias' for primary ventricular fibrillation were sought in the continuously recorded electrocardiograms of two groups—17 patients who developed primary ventricular fibrillation and 21 similar patients who did not. None had received antiarrhythmic therapy in the preceding 72 hours. Electrocardiographic analysis up to the twelfth hour after infarction was performed by three independent observers and employed a specially developed computer system. An R-on-T ventricular ectopic complex ($QR'/QT \leqslant 0{\cdot}85$) initiated all episodes of ventricular fibrillation but initiated only 1·5 per cent of episodes of ventricular tachycardia (4 of 265). The time course of R-on-T complexes differed from other ventricular arrhythmias including ventricular tachycardia, but closely paralleled the pattern of ventricular fibrillation. One or more R-on-T complex were recorded in 11 patients in each group. A mean prevalence of $>1/1000$ minutes of electrocardiogram occurred in eight patients with ventricular fibrillation and only one patient without ventricular fibrillation. In the 10 minutes before ventricular fibrillation the group incidence of R-on-T complex showed a striking increase.

The different time relation of R-on-T complexes and other ventricular arrhythmias and the prevalence and change of prevalence of R-on-T complexes, while not of practical value in predicting ventricular fibrillation, may contribute to our knowledge of the genesis of ventricular arrhythmias in acute myocardial infarction.

Relation of ventricular ectopic beat frequency to heart rate and everyday physical activity

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A method has been developed to study the effect of heart rate and of everyday physical activity on the ventricular ectopic beat frequency of patients monitored within six weeks of a myocardial infarction. The technique involves a computer analysis of 24-hour recordings of electrocardiogram and walking activity, and this study was based upon 49 such recordings, throughout all of which the ectopic beats were shown to be correctly identified by computer. A statistically significant relation between ectopic beat frequency and heart rate was shown in 39 (80%) of these recordings, with 24 having ectopic beats predominantly at the lower heart rates and 15 at the higher heart rates. Similarly, walking activity affected ectopic beat frequency in 14 (74%) of the 19 patients studied after discharge from hospital and the response to activity reflected the response to heart rate.

All the patients whose ectopic frequency increased at the higher heart rates were still alive one year after being studied and there was only one cardiac death among the patients with bradycardia-related ectopic beats. It therefore seems unlikely that either type of response of ectopic frequency to heart rate carries a particularly adverse prognosis.

Non-invasive assessment of diastolic function in hypertrophic cardiomyopathy on and off beta-adrenergic blocking drugs

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Beta-adrenergic blocking drugs in hypertrophic cardiomyopathy provide symptomatic relief but their effect on long-term prognosis is uncertain.

Thirty patients have been studied non-invasively by simultaneous recordings of echocardiogram, apex cardiogram, phonocardiogram, and electrocardiogram in order to assess diastolic abnormalities on and off oral beta-adrenergic blocking drugs. While on treatment all these patients had a mean dose of propranolol of 200 mg/day and the drug was stopped for one week before repeating the non-invasive studies for assessment off treatment. The following indices were studied; isovolumic relaxation period (A_2 to mitral valve opening), relaxation period (A_2 to 'O' point of apex cardiogram), peak left ventricular filling rate, and the duration of the rapid filling phase.

Prolonged isovolumic relaxation period and relaxation period reflect a reduced rate of fall of left ventricular pressure and in this study both isovolumic relaxation period and relaxation period were shortened in seven and prolonged in 10 patients. Eleven patients showed inconsistent results and two remained the same after propranolol. Most of the patients had a prolonged isovolumic relaxation period and relaxation period before propranolol. The alteration in the peak left ventricular filling rate and the duration of the rapid filling phase will be discussed.

It is concluded that the response of diastolic indices to beta-blocking drugs in hypertrophic cardiomyopathy is variable and that there is a group of patients who improved their diastolic function with beta-blocking drugs.

Changes in QRS amplitude during maximal exercise testing and relation to presence of coronary artery disease and left ventricular dysfunction

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To test the theory that QRS amplitude varies with change in ventricular volume, changes in R wave amplitude were measured sequentially during and after 12 lead maximal treadmill exercise in 62 patients with coronary artery disease and 14 normal subjects. At maximal exercise R wave amplitude increased in patients with coronary artery disease by 2·4 ($\pm$0·3) mm and in normal subjects decreased by 3·2 ($\pm$0·4) mm. In addition to differential amplitude changes recovery time was delayed in patients with coronary artery disease and left ventricular dysfunction, being 8·6 ($\pm$0·8) minutes in patients with three vessel coronary artery disease and left ventricular dyskinesis or akinesis, 3·9 ($\pm$0·7) minutes in patients with single vessel coronary artery disease, and 3·0 ($\pm$0·6) minutes in normal subjects. Increase in R wave amplitude was greatest in patients with akinesis or dyskinesis (3·5 $\pm$0·5 mm) compared with those with localised hypokinesis (2·3 $\pm$0·3 mm). Increase in R wave amplitude was an independent indicator of coronary artery disease with no correlation between regional R wave changes and areas of ischaemia on the 12 lead exercise electrocardiogram.

Changes in R wave amplitude and behaviour may distinguish patients with coronary artery disease from normals and provide a non-invasive marker of left ventricular dysfunction.

Further experience with anatomical correction of simple transposition of the great arteries

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Between 1975 and 1979, 33 patients underwent anatomical correction of transposition of the great arteries. Of these, 14 aged between 1 month and 2·5 years had simple transposition and underwent two-stage anatomical correction. After the first stage operation of pulmonary artery banding the peak systolic left ventricular pressure rose from 32 $\pm$12 to 80 $\pm$30 mmHg with no significant change in end-diastolic pressure or ejection fraction. This was associated with decrease in actual pulmonary flow, but no change in effective pulmonary flow.

Correction was performed four weeks to eight months (mean three months) later. There were four early deaths and no late deaths (follow-up from three to 36 months). After an initial period of cardiac failure the remaining 10 patients became asymptomatic with normal development. Rapid change in electrocardiogram, vectorcardiogram, and echocardiogram towards normality was observed. Repeat cardiac catheterisation was performed in seven, three weeks to two-and-a-half years after operation. Left ventricular ejection fraction was normal in all, mild mitral regurgitation was present in two investigated early, minimal aortic regurgitation in four, and moderate supravalvar pulmonary stenosis in one. The aortic and coronary anastomoses appeared to grow normally.

Pulmonary blood supply in pulmonary atresia with ventricular septal defect and multifocal pulmonary blood supply

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We have previously shown by examination of necropsy material in patients with pulmonary atresia and ventricular septal defect with multifocal pulmonary blood supply that the hypoplastic central pulmonary arteries frequently connect to a minority of the 19 broncopulmonary segments, and we suggested that this would limit the effectiveness for surgery. In order to see whether these findings were biased by virtue of the patients having died, we reviewed the pre- and postoperative angiocardiograms, including selective collateral injections, in nine patients who survived a shunt operation.

Central pulmonary arteries were present in six patients and absent in three. Insertion of a shunt to a central pulmonary artery in five cases improved perfusion in only six to seven segments of lung per case. In the remaining four cases, a shunt into an intrapulmonary artery, connected to a collateral, increased perfusion to three to eight segments of

each lung. Central and lobar pulmonary arteries appeared larger, but stenoses within and between these vessels were frequent and the peripheral vessels continued to appear abnormally small.

We conclude that in a high proportion of cases, the total pattern of pulmonary vascular supply will preclude satisfactory total repair unless present management techniques are improved.

Risk factors after balloon atrial septostomy in patients with complete transposition

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Despite balloon atrial septostomy, some patients with complete transposition of the great arteries die before reaching elective definitive surgery in the second six months of life. To discover why, we analysed the fate of 144 patients who had had balloon atrial septostomy since 1966, using a modified logrank survival test with multivariate capability. Patients were withdrawn alive on reaching definitive surgery.

The following largely independent factors were associated with a statistically significant ($P < 0.05$) excess mortality: pulmonary hypertension, the presence and size of ventricular septal defect or persistent ductus arteriosus, relative anaemia, absence of left ventricular outflow tract obstruction, low arterial oxygen saturation, aortic stenosis and coarctation, and balloon atrial septostomy between one week and one month of life.

Those of the above factors that can be determined at balloon atrial septostomy or routine three-month cardiac catheterisation were then introduced into discriminant function analysis on survival to six months. Hence the probability of any individual patient dying in the first six months was calculated, allowing for these factors. This prediction was correct in 72 per cent of the patients studied. By offering earlier definitive correction to patients thus identified as at high risk of premature death, it should prove possible to reduce overall mortality in transposition of the great arteries.

Atrial filling characteristics after the Mustard and Senning procedures: analysis by transcutaneous Doppler ultrasound

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Venous inflow correction of transposition of the great arteries involves construction of an interatrial baffle, which may consist either of living atrial tissue (Senning's operation) or pericardium/Dacron (Mustard's procedure).

To investigate whether this difference might affect atrial compliance and/or volume we recorded jugular venous waveforms in 24 patients operated on in Leiden, eight after Mustard's operation and 16 after Senning's. No patient had caval pathway obstruction.

Normal children display waveforms exhibiting two forward flow maxima of roughly equal height, one of atrial filling during ventricular systole and the other of ventricular filling.

After Mustard's operation, peak and total flow during atrial filling were reduced to a mean of 45 and 23 per cent of the ventricular filling phase, respectively. After Senning's operation these values were significantly higher, being 61 per cent ($P < 0.05$) and 44 per cent ($P < 0.001$), respectively.

Since the composition of the two groups was identical with regard to the mitral valve status and left ventricular pressure load, we conclude that this observed difference reflects a difference in atrial filling characteristics. The Senning operation appears to allow easier atrial filling because the living interatrial baffle is more compliant, and possibly also because the new systemic venous atrium is larger.

Surgical repair of common atrioventricular orifice without atrial septal defect in two patients

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Atrioventricular canal defects define a spectrum of cardiac malformations characterised by an aortic valve unwedged between the atrioventricular valves and a 'scooped-out' appearance of the interventricular septum. We distinguish between complete and partial forms on the basis of a common or partitioned atrioventricular orifice. Recently, we operated upon two cases of complete atrioventricular orifice without an ostium primum interatrial septal defect. In both, there was a common atrioventricular

orifice with the anterior common leaflet bridging the interventricular septum and attached to one papillary muscle in the right ventricle. There was a wide interventricular communication. The interatrial septum was well formed; its secure attachment to the superior edge of the anterior and posterior common leaflets prevented any interatrial communication. In both cases it was necessary to resect the inferior border of the interatrial septum in order to visualise the leftward components of the two bridging leaflets. The surgical repair of the thereby created complete atrioventricular canal Rastelli type C, was then carried out as usual by repair of the left atrioventricular valve and patch closure of the atrioventricular communication. Both patients are well three and five months after operation, though residual mitral regurgitation needed reoperation in one.

Common atrioventricular canal: late results after surgical repair

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Nine patients, aged 23 months to 39 years, operated on from 1964 to 1977 in the National Heart Hospital, left hospital after repair for common atrioventricular canal and have had complete follow-up with recatheterisation. Six had earlier palliative surgery: banding of the pulmonary artery (4), Waterston (1), Blalock anastomosis (1). The right ventricular pressure was systemic in seven and below in two with an unusual small canal defect.

During the follow-up period of two to 14 years, one died suddenly 14 years later having had no residual haemodynamic lesion but with changing bundle-branch block and prolongation of the PR interval with periods of asymptomatic bradycardia. The other eight had mitral regurgitation, mild in six, moderate in one, and severe in one whose calcified mitral valve was replaced eight years later.

The PR interval has increased in all but one who has had syncope associated with Wenckebach and ventricular tachycardia requiring pacing and propranolol 11 years after operation. No other rhythm disorder has occurred. The pulmonary artery pressure and resistance fell in all. One had a small ventricular septal defect. None had subaortic gradients.

The careful, long-term follow-up of patients with repaired common atrioventricular canal demonstrates good symptomatic and haemodynamic results but the conducting tissue and mitral valve function may continue to cause problems.

Interaction between cigarettes and propranolol in treatment of angina pectoris

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This study investigates whether cigarette smoking interferes with the medical management of angina pectoris. Ten patients with angina pectoris who smoked at least 10 cigarettes a day were studied by recording heart rate, blood pressure, and 16 lead praecordial electrocardiographic maps before and after a standardised maximal exercise test at the end of four randomly allocated two-week treatment periods. These periods were (a) on glyceryl trinitrate (GTN) off smoking, (b) on GTN while smoking, (c) on GTN and propranolol (240 mg daily) off smoking, and (d) on GTN and propranolol while smoking. Carboxyhaemoglobin was measured to ensure compliance. On GTN and off smoking the maximum heart rate achieved was 137 to 156 beats a minute, the maximum systolic blood pressure was 160 to 210 mmHg, and the maximum area and severity of ST segment changes was two to eight positions and 3 to 12 mm, respectively. An analysis of variance comparing the four groups showed that smoking was associated with a significantly higher heart rate, blood pressure, number of positions with ST segment depression, and total ST segment depression after exercise ($P < 0{\cdot}01$). This also occurred when patients were on propranolol ($P < 0{\cdot}01$). These results suggest that smoking aggravates the simple haemodynamic indices that are used to assess myocardial oxygen requrements and the exercise induced praecordial electrocardiographic signs of myocardial ischaemia. These effects were still evident after treatment with propranolol and represent a hindrance to the effective medical treatment of angina pectoris.

Propranolol in acute myocardial infarction

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Thirty patients with apparently uncomplicated

anterior myocardial infarction were considered for randomisation into a trial of oral propranolol 40 mg three times daily or placebo. The patients were studied within eight hours after the onset of their cardiac pain. A Swan-Ganz thermodilution catheter was used to measure both the pulmonary capillary wedge pressure and the cardiac output. Plasma catecholamines—noradrenaline and adrenaline, were measured. Five patients who had an 'inappropriate' tachycardia were found to have a significantly elevated pulmonary capillary wedge pressure of 21 ± 4 mmHg and were not given propranolol. In the propranolol group of patients heart rate fell from 87 ± 15 to 76 ± 9 beats/min ($P < 0{\cdot}01$) as did cardiac output from $4{\cdot}9 \pm 0{\cdot}7$ to $4{\cdot}5 \pm 0{\cdot}9$ l/min ($P < 0{\cdot}05$). There was no change in the wedge pressure 14 ± 2 and 13 ± 2 mmHg but mean systemic blood pressure fell from 103 ± 10 to 98 ± 10 mmHg. Plasma catecholamines, particularly noradrenaline, increased from $0{\cdot}26 \pm 0{\cdot}5$ to $0{\cdot}44 \pm 0{\cdot}7$ ng/ml. The haemodynamics did not change in the placebo group but plasma noradrenaline fell from $0{\cdot}44 \pm 0{\cdot}66$ to $0{\cdot}32 \pm 0{\cdot}56$ ng/ml. Four patients developed cardiac failure of whom three were receiving propranolol. These results suggest that 'inappropriate' tachycardia indicates left heart failure and that propranolol cannot safely be given to patients with an apparently uncomplicated anterior myocardial infarction. Propranolol also appears to increase plasma catecholamines in this group of patients.

Ergometrine induced 'angina'—a diagnostic pitfall

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We have reviewed patients diagnosed as having cardiac pain but normal coronary arteriograms and have fully reinvestigated those with persistent and convincing symptoms. A small group of patients has been identified who have typical cardiac pain which occurs at rest and with exertion, can be induced by coronary sinus pacing or by ergometrine, may be associated with T wave inversion, and is relieved by nitrites. These patients showed no evidence of any cardiac abnormality and again had structurally normal coronary arteriograms, transmyocardial arteriovenous lactate differences remaining normal with pacing or with ergometrine to the point of 'angina' in every case. Oesophageal manometry, however, showed that the ergometrine-induced 'angina' was associated with the development of oesophageal spasm. No oesophageal spasm was seen in these or other patients given ergometrine in the absence of pain.

This study shows that 'angina' induced by ergometrine cannot be regarded as necessarily indicating coronary artery spasm. The ergometrine test may be useful in establishing a positive diagnosis of oesophageal spasm in this diagnostically difficult group.

Verapamil in chronic stable angina—controlled study with quantified treadmill exercise and computer analysed ST segment changes

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The antianginal efficacy of verapamil hydrochloride in a dose of 120 mg three times daily was assessed by serial continuous multistage exercise and on-line computer assisted ST segment analysis. Twenty-seven patients with confirmed effort-induced angina were evaluated by a double-blind placebo controlled cross-over study. Maximal treadmill tests were performed before treatment, two weeks after placebo, and two and four weeks after 360 mg daily of verapamil. Indices included angina attacks, consumption of glyceryl trinitrate, anginal time (time taken to develop angina on treadmill testing), cumulative ST depression, time taken to develop 1 and 2 mm ST change, and ST indices (cumulative ST depression corrected to time of exercise, oxygen consumption, METS equivalent, and workload). Thirteen patients became angina free after two weeks and 15 angina free after four weeks treatment. The treadmill exercise time increased from $6{\cdot}6 \pm 0{\cdot}4$ minutes to $11{\cdot}0 \pm 1{\cdot}0$ minutes ($P < 0{\cdot}001$). Pronounced improvements were observed at statistically significant levels ($P < 0{\cdot}001$) in trinitrin consumption, 1 mm time, 2 mm time, double product, heart rate response to exercise, cumulative ST depression, and ST units. Side effects consisted of constipation in seven, prolonged PR interval in two, and headache in one. None of the patients developed signs of cardiac failure. These results indicate that verapamil in a dose of 360 mg daily is a very powerful antianginal agent free from troublesome side effects.

Coronary haemodynamic effects of nifedipine: comparison with glyceryl trinitrate

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To determine the coronary haemodynamic effects of nifedipine, values for myocardial blood flow (coronary sinus flow—CSF), myocardial oxygen extraction (O_2 extr), and myocardial oxygen consumption (MVO_2) were obtained in sinus rhythm (SR) and during rapid atrial pacing (RAP) at rates insufficient to produce angina in 11 patients undergoing cardiac catheterisation for suspected coronary artery disease. In the patients with demonstrated coronary artery disease rapid atrial pacing rate was increased to produce angina. Measurements were repeated after 20 mg nifedipine sublingually. Results were compared with a group of patients who received 0·5 mg glyceryl trinitrate (GTN) sublingually.

In sinus rhythm, nifedipine reduced mean O_2 extr from 62 to 54 per cent (13%, $P < 0.02$) with no significant change in CSF or MVO_2. GTN reduced mean CSF from 171 to 115 ml/min (33%, $P < 0.02$) and MVO_2 from 19·9 to 12·3 ml/min (38%, $P < 0.01$), with no significant change in O_2 extr.

During rapid atrial pacing, nifedipine reduced mean O_2 extr from 59 to 54 per cent (8%, $P < 0.05$), with no significant change in CSF or MVO_2. GTN reduced mean CSF from 207 to 168 ml/min (19%, $P < 0.05$) and MVO_2 from 24 to 18·2 ml/min (24%, $P < 0.05$) with no significant change in O_2 extr.

A major effect of nifedipine was coronary vasodilatation whereas GTN reduced MVO_2 with no apparent coronary vasodilator effect.

Both drugs were effective in relieving pacing-induced angina, probably by different mechanisms.

M-mode subxiphoid echocardiographic scan in analysis of atrioventricular junction

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Analysis of the atrioventricular junction is important for sequential diagnosis in congenital heart disease. It is necessary to know (i) if one or both atria connect to the ventricular mass; (ii) if both connect, whether the ventricular inlets are separated; (iii) how many valves guard the junction; and (iv) the relations of these valves to the inlet ventricular septum. The usefulness of parasternal M-mode echocardiography is limited because of difficulty in profiling the inlet septum. Theoretically, a subxiphoid approach should provide this information. This study assessed its practical advantages and developed a composite subxiphoid scan in which the transducer beam is initially directed from the subxiphoid notch to cross right ventricular cavity, right atrioventricular orifice, and aorta. With subsequent sinistroposterior beam angulation the relations of right aortic wall and atrioventricular valves to the inlet septum are shown.

The scan was assessed in 30 normal children, 47 with atrioventricular defects and nine with univentricular hearts of left ventricular type, three with double inlet, and six with absent right atrioventricular connection. In all abnormal hearts the scan accurately predicted the atrioventricular junction anatomy found at surgery or angiography, suggesting that the subxiphoid scan is the technique of choice when studying the inlet septum.

Ventricular morphology—two-dimensional echocardiographic recognition in complex congenital heart disease

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Definition of ventricular morphology and their relations is mandatory in the diagnosis of complex congenital heart disease. Based on experiences in over 1000 studies of patients with normal cardiac connections, two-dimensional echocardiographic criteria for right (RV) versus left ventricular (LV) morphology were compared in 16 patients, all of whom had two ventricular chambers, biventricular angiography demonstrating transposition of the great arteries (TGA) in three, corrected TGA in 10, anatomically corrected malposition in one, and a 'criss-cross' heart in two. Criteria used for RV morphology were: insertion of chordae tendineae into interventricular septum; the presence of an infundibulum; the presence of trabeculated endocardium and a moderator band. Criteria for LV morphology were: the presence of two discrete papillary muscle groups and a smooth endocardium. Because of the invariable association of the AV connection with the respective ventricle attention was given to AV valve morphology, its attachment to the interventricular septum, and its cusp number.

In all patients with a posterior LV, two papillary muscle groups were seen. These were also observed in the anterior morphological LV in five of the 10

patients with corrected TGA, though the posterior ventricle was correctly identified as having RV morphology in all 10 patients by chordae inserting into the interventricular septum. Identification of infundibulum, the appearance of endocardium, and the presence of a moderator band were not useful as criteria. Correct identification of AV valve morphology was made in all 16 patients studied. Though the ventricular insertion of chordae may recognise the RV and LV, correct prediction of ventricular morphology was best achieved by identifying the associated AV valve.

Diagnostic accuracy of two-dimensional echocardiography in cyanosed infant

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Real-time two-dimensional echocardiography (60° mechanical sector scanner) has been applied to the preliminary assessment of a consecutive series of 59 infants (ages 14 hours to 6 months, weights 2·1 to 5·8 kg) suspected of having congenital heart disease and subjected to cardiac catheterisation. The basic diagnosis was correctly established with two-dimensional echocardiography in all those with transposition of the great arteries (16), pulmonary valve atresia or severe stenosis (5), left heart hypoplasia (4), Fallot's tetralogy or Fallot-type pulmonary atresia (5), atrioventricular canal (3), and truncus arteriosus (2). In six of seven infants with single ventricle the anatomy was correctly ascertained, and in four patients with complex anomalies the echocardiogram gave useful information though not the full diagnosis. The echocardiogram was misinterpreted in one infant with severe pulmonary hypertension and in one with total anomalous pulmonary venous drainage (TAPVD). No two-dimensional echocardiographic abnormality was found in the other patients with pulmonary hypertension (3) or in patients with respiratory disease (2). TAPVD was strongly suspected but not diagnosed with certainty in four. In two, the quality of the two-dimensional image was inadequate for diagnostic assessment. Two-dimensional echocardiography provides a rapid anatomical diagnosis in most cyanotic infants. With further experience of the technique even more reliable diagnostic interpretations are probable.

Long-term oral prostaglandin E_2 in ductus dependent pulmonary circulation

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Short-term intravascular prostaglandin E_1 or E_2 (PGE_1 or PGE_2) effectively maintains ductus arteriosus patency in neonates whose pulmonary circulation is ductus dependent. We have evaluated oral PGE_2 administration in seven infants with pulmonary atresia or tricuspid atresia, in five of whom the pulmonary arteries were initially considered too small for anastomotic operations. PGE_2 was given orally, 30 to 250 μg hourly to four-hourly, for six days to four months with repeated measurements of arterial oxygen tension (PaO_2) or saturation (SaO_2). Discontinuing treatment for two to eight hours in individual cases caused PaO_2 or SaO_2 to fall by 25 to 35 per cent of the values achieved on treatment; they rose again as early as 15 minutes after an oral dose. These changes were closely related to plasma PGE_2 concentrations measured by radioimmunoassay. Satisfactory levels of SaO_2 ($>65\%$) could be maintained only while treatment continued. Three infants, recatheterised four to 10 weeks after starting PGE_2, showed growth of pulmonary arteries. Aortopulmonary shunts, attempted in five infants, have been successful in four. The effectiveness and relative simplicity of oral PGE_2 treatment suggest that it may supersede intravascular infusions for both short- and long-term purposes.

Fetal tachycardia

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The incidence of fetal arrhythmias is not known but the few reports suggest that it is seldom recognised. We are reporting three infants who were delivered by emergency caesarean section because they had persistent tachycardia. Two were cyanosed at birth, case 1 because of transposition of the great arteries and case 3 because of persistence of the fetal circulation. Cases 2 and 3 did not have a congenital cardiac defect.

The three infants presented with atrial flutter. The first infant could not be converted to sinus rhythm by drug therapy or DC conversion and died at one week from aspiration, despite a successful atrial septostomy. The second infant was also refractory to treatment but well compensated and reverted spontaneously to sinus rhythm by 2 months of age. The third patient was easily converted to sinus rhythm by DC countershock.

Necropsy on case 1 showed a hypoplastic sinus node represented by a small area of cells with increased fibrous tissue. These findings suggested that this infant could not have reverted to sinus rhythm while the other two infants probably represented delayed maturation of the sinus node.

Our findings show that persistent fetal tachycardia may be the result of an abnormal rhythm and that histological examination of the conducting tissue in other fatal cases may be helpful in understanding the mechanism of tachycardias in the fetus and neonate.

Quantitative dynamic study of distribution of thallium-201 in normally perfused, ischaemic, and infarcted myocardium

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Time-dependent regional distribution of thallium-201 was measured by serial imaging for 24 hours after injection during maximal exercise. Normal myocardium showed peak activity 30 minutes after injection followed by an exponential decline independent of decay of the isotope. Infarcts reached peak activity at the same time, but never exceeded 50 per cent of the highest activity in normal myocardium.

Counts recorded represent the activity of a cylinder formed by two walls and the cavity of the ventricle. The activity is a composite of the infarcted zone and overlying, or underlying, normal myocardium. Subtraction of the normal activity curve from the infarct curve shows that all activity is obtained from normally perfused muscle; the infarct contributes only as an inactive 'window'.

Ischaemic myocardium showed delayed uptake reaching a peak one-and-a-half hours after injection and maintaining that level of activity until equal to the falling activity of adjacent normal muscle. If the contribution of superimposed normal myocardium is subtracted, ischaemic muscle is shown to accumulate thallium-201 for four to six hours before reaching a peak and then declining at a similar rate to normal muscle.

The findings are important in interpreting thallium-201 scintigrams and may facilitate the identification of preclinical ischaemia.

ECAT imaging in acute myocardial infarction

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Recent developments in emission computed tomography (ECAT) allow cross-sectional three-dimensional imaging of the heart. This overcomes the superimposition of anatomical 'non-interest' regions of conventional scanning. The images reflect quantitatively the distribution of the radioactive indicator concentration in the myocardium and thus provide a potential means of quantifying regional myocardial perfusion and necrosis.

We have evaluated the suitability of ^{99m}Tc-IDP imaging in acute infarction with a transaxial emission tomography device (slice time five minutes) and compared it with standard scintigraphy in 20 patients. Though of similar sensitivity, the tomographic approach in the ECAT sections offers an opportunity for quantification of infarct size and this is the significant clinical potential of the technique. In addition, the difficult diagnosis of subendocardial infarction appears to benefit from the improved contrast in the ECAT sections.

Correlation of thallium and technetium scintigraphy: method for assessing within-patient interventions on infarct size

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It is established that technetium-99^{m} (^{99}Tc^{m}) phosphate compounds can be used to estimate the size of an infarct some days after its onset, and that thallium-201 (^{201}Tl) scintigraphy can show perfusion defects demonstrable early in the evolution of an infarct. ^{201}Tl imaging was performed two-and-a-half to eight (mean 5·3) hours after the onset of symptoms in 17 patients with typical history of cardiac pain >1 hour, ST elevation >5 mm, and no evidence of old infarction. ^{99}Tc^{m} polyphosphate imaging was performed in an identical left anterior oblique projection in all patients 36 to 48 hours after the onset of symptoms. The area of the ^{201}Tl-defined perfusion defect and that of the ^{99}Tc^{m} polyphosphate-defined infarct were computed after background subtraction. Clearly-defined areas both of transmural ^{201}Tl perfusion defect and Tc polyphosphate uptake were seen in 16 of the 17 patients, and these areas showed good correlation ($r=0{\cdot}893$, $P<0{\cdot}001$). The other patient showed a transmural defect of ^{201}Tl activity, was treated three hours after the onset of pain with glyceryl

trinitrate, and subsequently showed no clearly-defined region of Tc polyphosphate uptake.

This study provides the basis of a method suitable for within-patient testing of early interventions on infarct prevention or limitation.

Exercise first pass radionuclide ventriculography in assessment of coronary artery bypass surgery

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Fourteen patients underwent exercise first pass radionuclide ventriculography (RV) before and after coronary artery bypass surgery. Twelve were angina-free on maximal exercise postoperatively and two developed angina.

In the angina-free group there was no significant change in resting left ventricular ejection fraction after surgery, but mean exercise ejection fraction rose significantly from 55·6 to 63·2 ($P < 0·025$). Preoperatively, mean ejection fraction fell on exercise to angina from 62·8 to 56·1 per cent ($P < 0·05$). On exercise after operation there was no change in ejection fraction compared with the resting value, which represents a significant improvement on the preoperative result ($P < 0·05$). In the two patients who developed angina after surgery there was a mean fall in ejection fraction on exercise of 9 per cent.

Wall motion abnormalities were assessed by a hemiaxial method. In the angina-free group, all 14 zones of regional wall motion abnormalities unmasked by exercise before operation were not present postoperatively. Both regions of wall motion abnormalities induced by exercise in the patients with recurrent angina were also seen preoperatively. Coronary artery bypass graft surgery results in an improvement in regional and global ventricular function during exercise. First pass RV should prove valuable in the assessment of recurrent angina after surgery.

Vagotonic sinus bradycardia and paroxysmal atrial fibrillation

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In some patients with lone atrial fibrillation, intense bradycardia and associated atrial arrhythmias may be seen during natural periods of dominant vagal tone.

Nine patients, all male (ages 14 to 52) and free of other cardiac abnormalities gave a history of bradycardia and palpitation during sleep or relaxation. Paroxysmal sinus bradycardia (45/min or less) was seen on routine electrocardiogram or 24-hour ambulatory electocardiographic monitoring. Routine electrocardiograms were normal in all though in one P wave morphology was consistent with minor atrial conduction disturbance. Electrophysiological tests were normal in all but one patient in whom sinus node recovery was prolonged (3·5 s) only at the slowest pacing rate tested. On exercise testing there was normal sinus acceleration and prompt, often extreme, bradycardia in the immediate resting phase. Similarly, paroxysmal atrial fibrillation occurred during the rest after exercise or at other times of dominant vagal tone. Treatment with disopyramide or quinidine was successful in one of five patients, while amiodarone was effective in all three to whom it was given.

Patients with unduly pronounced effects of vagal stimulation may have paroxysmal atrial fibrillation and masquerade as sinus node disease. While pacing may help such patients symptomatically there was no evidence of organic sinuatrial disease in this series.

Do antiarrhythmic drugs depress sinus node function in patients with sick sinus syndrome?

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The potential need for pacing has been examined in 39 patients with sick sinus syndrome requiring either digoxin, propranolol, verapamil, or disopyramide for their associated tachycardias. Assessment with an acute electropharmacological pacing study for effects of these drugs on basic sinus cycle length (SCL) and on sinus node recovery time (SNRT); and with 24-hour electrocardiographic monitoring for effects of oral treatment on lowest heart rate is shown in the Table.

All four antiarrhythmic drugs depress sinus node function, though this is least pronounced with digoxin. The acute study is unreliable in predicting dangerous sinus node depression with oral treatment which should be started in hospital with temporary pacemaker cover (rate 30 bpm) and with monitoring to assess the need for permanent pacemaker implantation.

Drug	*Acute study* *SCL (ms)*	*SNRT (ms)*	*Comment*	*Chronic oral study* *Lowest mean HR (bpm)*	*Patients with transient brady-cardia (30 bpm)*
Control	1050 ±112	1510 ±315		53 ±8	
Propranolol (n=9)	1210 ±170 (P=0·005)	2140 ±510 (P=0·001)		45 ±9 (P=0·005)	3
Control	907 ±230	1570 ±510		68·7 ±9·3	
Verapamil (n=8)	1270 ±110 (P=0·005)	1860 > 425 (P=0·005)		57·8 ±7·5 (P=0·01)	2
Control	987 ±110	1610 ±210		55 ±4	
Digoxin (n=12)	1070 ±205 (P=0·05)	1790 ±510 (P0·01)		51 ±7 (P=0·1)	1
Control	905 ±190	1740 ±310	× 1 sinus arrest > 2 min	63·7 ±11i	
Diso-pyramide (n=10)	801 ±210 (P=0·01)	2140 ±620 (P=0·005)		59·5 ±9 (P=0·01)	4

Unexplained syncope resulting from masked sinus node dysfunction

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Electrophysiological investigation has not proved rewarding in the diagnosis of sinus node abnormalities not otherwise detected, probably because of alteration in autonomic tone induced by anxiety and by rapid atrial pacing.

Twenty-three patients with unexplained syncope or near syncope were therefore investigated by measurement of sinus node recovery time (SNRT) (2 min atrial pacing at 100 and 180/min), calculated sinus atrial conduction time (SACT), and intrinsic heart rate, before and after autonomic blockade with atropine (0·04 mg/kg) and propranolol (0·2 mg/kg). Sixteen asymptomatic controls with normal coronary arteries, haemodynamics, and conduction systems were investigated for ST-T changes only.

Intrinsic heart rate was normal in all controls but abnormal in 11 of 23 patients. SACT was normal and unaffected by autonomic blockade in both groups, as was SNRT after pacing at 100/min. SNRT was also normal before blockade after pacing at 180/min in both groups but, after blockade, though SNRT was normal in controls, it was prolonged abnormally to 158 to 325 per cent of previous sinus cycle length in nine of 23 symptomatic patients; six of these nine had a slow intrinsic heart rate, and permanent pacing relieved syncope in five. In 14 of 23 symptomatic patients with normal SNRT, only two had an abnormal intrinsic heart rate, and pacing in one did not help.

Autonomic blockade may thus increase the sensitivity of electrophysiological investigation for sinus node disorders and may unmask abnormalities of sinus node automaticity too infrequently manifest for detection by ambulatory electrocardiogram.

Spectrum of AV conduction abnormalities and tachycardias associated with anomalous Mahaim conduction

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Of 67 patients investigated for paroxysmal tachycardias associated with the WPW syndrome, six had electrophysiological evidence of anomalous nodoventricular conduction. The patients (4 female, 2 male) were aged between 13 and 58 years. All had WPW type B QRS complexes but in two patients this was intermittent. In four, regular paroxysmal tachycardias with broad QRS complexes had been documented before study. Electrophysiological studies revealed the following results: (1) short HV interval during sinus rhythm (three cases); (2) prolongation of the AH and AV intervals, with increasingly premature atrial beats in five patients. In four patients this was accompanied by increasing pre-excitation. Pre-excitation within a fixed zone of coupling intervals of premature atrial beats occurred in one patient; (3) sudden prolongation of the AH (or AV) interval at a critical coupling interval of premature atrial beats in three patients. This was accompanied by loss of pre-excitation in one patient. The sudden jump in AH was associated with an atrial echo or tachycardia in two. Atrial echo occurred in two other patients either after a sudden jump or in the absence of this phenomenon; (4) pre-excited QRS complexes during tachycardia were seen in four patients. In two of these the His spike was clearly visible with an HV interval of 0 and −25, respectively; (5) the regular tachycardias were intranodal in origin; (6) during atrial pacing, Wenckebach periods terminated by simultaneous block in both AV node and nodoventricular pathway occurred in four patients; (7) in one patient profound bradycardia in response to atrial fibrillation was noted. During this rhythm the QRS complexes were uniformly pre-excited.

The observations in this series suggest that the conduction properties of nodoventricular pathways are determined by an intimate relation of the nodoventricular pathway to the AV node and the characteristics of intranodal conduction.

Bayesian analysis of stress thallium-201 scintigraphy

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Test characteristics, true positive ratio and true negative ratio were obtained for stress thallium-201 (^{201}Tl) scintigraphy (MPI) by comparison with coronary arteriography in 100 patients. Arteriography showed significant coronary heart disease in 60 patients and normal vessels in 40 patients.

True positive ratio for MPI was 0·90 and for true negative ratio was 0·88. Using Bayesian analysis, the probability of having coronary heart disease (pCHD) for a positive MPI result and negative MPI result was calculated for prevalences of coronary heart disease ranging from 1 to 99 per cent.

Discrimination was greatest where pretest pCHD lay between 30 and 70 per cent. At pretest pCHD of 52 per cent discrimination was maximum with pCHD with positive MPI being 89 per cent and with negative MPI being 11 per cent. At pretest pCHD of 20 per cent, pCHD with positive MPI fell to 64 per cent and with a negative MPI was 3 per cent. Below 20 per cent pretest pCHD, false positive tests increased sharply. At pretest pCHD of 80 per cent, pCHD with positive MPI was 97 per cent and with negative MPI 36 per cent. Above 80 per cent pretest pCHD, false negative tests increased sharply.

These results suggest that MPI would be an inappropriate test where pCHD is low, for example, population screening, but may be useful where clinical pCHD lies between 30 and 70 per cent.

Clinical implication of normal exercise ECG and thallium-201 scintigram

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Exercise thallium-201 scintigraphy (MPI) provides a non-invasive technique for identifying the presence of coronary heart disease in patients presenting with chest pain. However, the ability of a normal study to exclude coronary heart disease is not clear. Maximal exercise testing (MET) and MPI were performed in 115 patients having diagnostic catheterisation. Image data were interpreted by computer analysis and the results compared with arteriography. In 69 patients, arteriography demonstrated significant coronary heart disease ($\geqslant$50% vessel occlusion); 46 patients had normal vessels. The proportion of misclassification for normal MET, normal MPI, and combined normal MET and normal MPI was calculated.

In 46 patients with normal vessels, MET was normal in 34, MPI was normal in 39, and MET + MPI normal in 31. In 69 patients with coronary heart disease, MET was normal in 19 and MPI was normal in seven. Combined normal MET and normal MPI was not encountered in patients with coronary heart disease. The proportion of misclassification for normal MET was 36 per cent (19/53), for normal MPI was 17 per cent (7/46), and for combined normal MET and normal MPI 0 per cent (0/31). Thus, the combination of normal maximal exercise testing and normal ^{201}Tl scintigraphy occurred in 67 per cent (31/46) of patients with normal vessels and reliably excluded coronary heart disease.

What is best stress test to detect coronary artery disease using technetium-99^{m} gated cardiac blood pool scintigraphy (^{99}Tc^{m} GBPS)?

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Investigations were carried out on 34 normotensive patients (15 normals, 19 coronary artery disease) with normal or only slightly impaired left ventricular function using ^{99}Tc^{m} GBPS at rest and during peripheral cold stimulation, isometric handgrip, and dynamic supine exercise to determine the relative potency of these interventions to provoke an abnormal left ventricular ejection fraction (LVEF) response (>10% fall) in patients with coronary artery disease. The average double product (heart rate $\times$ mean blood pressure) at rest was 7319 mmHg/min and increased with cold stimulation, isometric handgrip, and dynamic supine exercise to 11 159, 12 495, and 16 611 mmHg/min, respectively.

Patients with normal hearts had no significant change or a slight increase in LVEF with each intervention whereas patients with coronary artery disease had a significant fall in LVEF with cold stimulation ($P < 0·001$) and dynamic supine exercise ($P < 0·002$) but not with isometric handgrip (NS). Cold stimulation identified 15 of 19 (79%) coronary artery disease patients whereas dynamic supine

exercise and isometric handgrip identified only six of 15 patients (40%) and two of 12 patients (17%), respectively. In comparison, single-lead V5 exercise electrocardiography detected only five of 19 (26%) patients with coronary artery disease.

We conclude that cold stimulation, despite evoking a smaller increment in double product, is a more convenient and sensitive stress intervention than dynamic supine exercise or isometric handgrip to detect coronary artery disease patients with $^{99}Tc^{m}$ GBPS. This may be the result of cold stimulation mediated increase in coronary vascular resistance in coronary artery disease patients not caused by dynamic supine exercise or isometric handgrip.

Comparison of thallium-201 (^{201}Tl) myocardial imaging after coronary vasodilatation with dipyridamole and after maximal treadmill exercise

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Traditional ^{201}Tl myocardial imaging is performed after maximal exercise. With many patients this is difficult because of locomotor disability, poor motivation, easy fatigue, or drug effects. Coronary vasodilatation with intravenous dipyridamole has been suggested as an alternative to exercise in demonstrating defects in myocardial perfusion.

Fifteen patients having coronary arteriography underwent ^{201}Tl imaging on two separate occasions: after intravenous dipyridamole and after exercise. Early and late images were recorded. The images were assessed independently by three experienced observers. There was good interobserver agreement, and any discrepencies were resolved by combined viewing of the images. Images were examined for diagnostic quality and for defects in activity. Myocardial/background counts were assessed by computer analysis. Correlations with the coronary arteriograms were made.

In 11 patients (73%) all images were of satisfactory diagnostic quality. In no patients were both early and late dipyridamole studies unsatisfactory. In nine patients both exercise and dipyridamole images showed defects. In five of these the late exercise image showed redistribution of activity into these defects, while redistribution after dipyridamole was demonstrated in three. In the remaining six patients there were no defects on either image.

A minor (10%) fall in blood pressure occurred after dipyridamole, and angina and ST segment depression were seen less commonly than with exercise.

Intravenous dipyridamole is a reliable alternative to exercise in ^{201}Tl imaging, and in certain patients may be the technique of choice.

Dipyridamole compared with exercise for thallium scanning

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Intravenous dipyridamole as a coronary vasodilator was compared with exercise as a method of enhancing perfusion defects for detection by thallium-201 scintiscans. Scans from 20 patients were projected individually in random order and comparisons were made only after defects had been scored by three observers. The myocardial outline was divided into two segments in PA and lateral projections and three in the LAO 45 projection. Defects were identified in 65 segments using dipyridamole and 71 segments after exercise, but only 92 of the 140 segments were scored identically as normal or abnormal by the two methods. A fair correlation was obtained when the extent of myocardial defects was quantified irrespective of site using a grid system in four projections ($r=0{\cdot}688$, $P<0{\cdot}001$). Fifteen of the series had coronary angiograms. Dipyridamole predicted the site of obstructive lesions as accurately as exercise. No important adverse effects were observed from the drug in this small series. Intravenous dipyridamole can reasonably be used instead of exercise for thallium-201 scans but there are no clear-cut advantages unless exercise testing is impracticable.

Thallium-201 myocardial imaging in triple vessel disease

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Doubts have been cast about the value of ^{201}Tl myocardial imaging because of the reported incidence of normal scans in patients with proven coronary artery disease. Our study suggests that

the incidence of false negative scans is lower than previously reported.

Rest exercise ^{201}Tl imaging was performed on 70 patients who were undergoing routine coronary angiography. The scans were computer processed, code-marked, and reported from Polaroid colour photographs without knowledge of the clinical details. A perfusion defect was considered to be present where there was a discrete zone of reduced tracer activity ($\geqslant$20%).

Coronary angiograms were reported without knowledge of the scintigraphic findings. A vessel was considered diseased when a lesion was seen $\geqslant$70 per cent of the vessel lumen. Of the 70 patients, 21 were found to have triple vessel disease (left main coronary lesions were taken to indicate involvement of the left anterior descending and circumflex arteries). Of the 21 patients, 20 also had technically satisfactory rest exercise scans.

Nineteen of these 20 (95%) showed exercise induced perfusion defects, so that in this study a false negative occurred in only one patient (5%). For triple vessel disease this is a higher sensitivity than previously reported, enhancing the value of ^{201}Tl imaging as a non-invasive test for coronary artery disease.

Patterns of disturbed myocardial perfusion in patients with coronary artery disease

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Fifty patients with anginal chest pain were studied to determine whether different patterns of disturbed regional myocardial perfusion with stress are useful as a physiological measure of severity of coronary artery stenoses. All the patients underwent a standardised maximum exercise test with electrocardiography, left ventricular and coronary angiography, and measurement of regional perfusion using Krypton—81m and a standardised atrial pacing test. Eleven patients with normal coronary arteries and negative exercise tests demonstrated uniform regional myocardial perfusion with pacing. In 40 of the 50 patients with significant ($\geqslant$70% stenosis) coronary artery disease and positive exercise tests, atrial pacing produced both regional increases and regional decreases of myocardial perfusion. Analysis of regional perfusion in the segments showing decreases in perfusion showed three results. *Group 1:* regional perfusion in 10 patients increased significantly (by $21 \pm 7{\cdot}0$%, $P < 0{\cdot}01$) for seven to 10 minutes of atrial pacing and then decreased to 61 ± 9 per cent below control. This group achieved 30 000 to 45 000 Joules in the exercise test.

Group 2: regional perfusion in 12 patients remained stable for four to nine minutes of atrial pacing and then decreased to $69 \pm 11{\cdot}0$ per cent ($P < 0{\cdot}01$) below control. This group achieved 26 000 to 34 000 Joules in the exercise test. *Group 3:* regional perfusion in 17 patients showed an immediate decrease in myocardial perfusion to the affected segment with the start of atrial pacing, reaching 81 ± 14 per cent below control ($P < 0{\cdot}01$). These patients achieved 7000 to 22 000 Joules in the exercise test. In all these 40 patients ST segment depression and chest pain occurred at 140 ± 17 s and 240 ± 70 s, respectively, only after regional perfusion had decreased below control values. These patients with coronary artery disease showed a close relation between the three different patterns of disturbed perfusion with stress, the workload achieved in the exercise tests, and the timing of ST segment depression. These different patterns of myocardial perfusion help our understanding of coronary stenosis.

Beta adrenergic control of coronary artery smooth muscle

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The extent to which coronary artery spasm contributes to the spectrum of coronary disease remains uncertain and there is some evidence that beta-blocking drugs may be detrimental in coronary spasm. We studied the effect of various sympathetic agonists and antagonists on the mechanical responses of isolated coronary strips from sheep hearts. The $beta_2$ agonist salbutamol exerted no effect up to 10^{-6} M, whereas the $beta_1$ and $beta_2$ agonist isoprenaline induced relaxation at 10^{-11} M. The relaxing effect of isoprenaline was competitively antagonised in a dose-related manner by the beta-blocking agents propranolol, acebutalol, practolol, and oxprenolol in concentrations of 10^{-7}, 10^{-7}, 10^{-5}, and 10^{-7} M, respectively. Though these drugs were used in approximately equipotent beta-blocking doses, the isoprenaline dose-response curves were shifted respectively $\times 150$, $\times 65$, $\times 25$, and $\times 15$, indicating that propranolol blocked the isoprenaline-induced relaxation 10 times more than oxprenolol.

This study shows that the relaxation of coronary artery smooth muscle was mediated by $beta_1$

receptors, which contrasts with $beta_2$-mediated dilatation of other vascular beds. The possession of $beta_2$-blocking properties (non-cardioselectivity) was thus irrelevant to the effect of beta-blocking drugs on coronary artery tone in these experiments. The possession of partial agonist activity appeared to minimise their antagonism of $beta_1$-mediated dilatation. These findings may be relevant to the management of coronary disease in man.

Haemodynamic effects of beta-blocking drugs in non-obstructive hypertrophic cardiomyopathy

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Beta adrenergic blocking drugs in hypertrophic cardiomyopathy provide symptomatic relief and control of atrial fibrillation but their effect on long-term prognosis is uncertain, and their acute haemodynamic effects on diastole are still unclear.

Four patients have been studied by catheter-tip manometry before and after intravenous administration of 0·05 mg/kg metoprolol. This dose did not alter average resting heart rate. Diastolic pressure volume relations were calculated from continuous pressure measurement during left ventriculography. The effect of metoprolol on end-diastolic pressure was variable, but in all patients the early diastolic pressure dip was higher after metoprolol ($+5·8$, $P < 0·05$) and the end-diastolic volumes were increased ($+5·6$, $P < 0·05$). There was an insignificant fall in the ejection fraction. Simultaneous recording of the echocardiogram, apex cardiogram, phonocardiogram/electrocardiogram before and after oral beta-blocking drugs demonstrated no consistent shortening of isovolumic relaxation time, total relaxation period, or alteration of the peak left ventricular filling rate and duration of the rapid filling phase.

Retrospective review of the clinical records of patients with hypertrophic cardiomyopathy from 1973 to 1975 showed seven patients in whom intravenous administration of practolol 0·1 mg/kg did not lower end-diastolic pressure significantly and variably affected end-diastolic volume.

It is concluded that there is insufficient evidence of improvement in the majority of patients with HOCM after beta-blocking drugs to justify their administration routinely to these patients. Specific haemodynamic or arrhythmic features which can be improved by beta-blockers must be identified in individual patients.

Effects of prostacyclin on cardiac function in man

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Prostacyclin (PGI_2), a potent endogenous inhibitor of platelet aggregation, has also been shown to have vasodilator activity. We have intestigated the effects of infused prostacyclin on blood pressure and cardiac function, by non-invasive methods.

Six normal male volunteers were infused with PGI_2 at rates of 1, 2, 4, and 8 ng/kg per min, each increment lasting 20 minutes. Before infusion and during each increment the following measurements were made: (i) blood pressure, (ii) heart rate, (iii) left ventricular echocardiographic dimensions, (iv) systolic time intervals, and (v) percentage maximum adenosine diphosphate (ADP) induced platelet aggregation.

All results refer to differences between the control period and the final increment, and are the mean value $\pm$SEM. Diastolic blood pressure fell from 62 ± 3 mmHg to 53 ± 3 mmHg ($P < 0·02$), and heart rate rose from 62 ± 1 beat/min to 80 ± 1 beat/min ($P < 0·001$). Left ventricular end-diastolic dimensions fell from $50·2 \pm 0·31$ mm to $44·3 \pm 0·27$ mm ($P < 0·001$) and end-systolic dimensions from $30·0 \pm 0·27$ mm to $26·8 \pm 0·21$ mm ($P < 0·02$). Fractional shortening and velocity of circumfererential fibre shortening remained unchanged. The mean dose ratio of maximum ADP induced platelet aggregation was shifted to the right during PGI_2 at 4 (1·71; $P < 0·01$) and 8 (2·38; $P < 0·001$) ng/kg per min.

We conclude that PGI_2 causes a modest decline in diastolic blood pressure and increase in heart rate at rates which inhibit platelet aggregation in man. The reductions in end-diastolic and end-systolic dimensions occurring after PGI_2 suggest both arteriolar and venular dilatation, but there was no change in echo ejection phase indices.

Myocardial histamine type 2 receptors

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We have studied the effects of myocardial (H_2) receptor stimulation in man, using M-mode echocardiography to measure left ventricular dimensions. The effects of histamine infusion were compared with those of sodium nitroprusside

which has no direct cardiac effects, but which, like histamine, is a potent vasodilator. Both drugs were infused at rates which caused similar falls in diastolic blood pressure, to allow for changes in left ventricular dimensions directly and reflexly attributable to vasodilatation.

A single blind, randomised, crossover study was performed in six normal men. They received 4×20 minute intravenous infusions: (i) normal saline control, (ii) mepyramine 0·05 mg/kg per min (an H_1 receptor antagonist), (iii) histamine or sodium nitroprusside to produce a 15 mmHg fall in diastolic blood pressure (mean infusion rates were 1·5 and 0·6 μg/kg per min), (iv) with histamine or sodium nitroprusside continuing, cimetidine (an H_2 receptor antagonist) in an IV bolus of 100 mg, followed by 0·2 mg/kg per min.

Mepyramine caused no significant haemodynamic effects. Plasma noradrenaline rose from $0{\cdot}21 \pm 0{\cdot}04$ to $0{\cdot}30 \pm 0{\cdot}02$ ng/ml after histamine, and from $0{\cdot}22 \pm 0{\cdot}03$ to $0{\cdot}31 \pm 0{\cdot}07$ ng/ml after sodium nitroprusside, suggesting similar activation of the baroreflex. Plasma adrenaline was not significantly increased after either drug. Heart rate increased by 12 ± 4 beats/min after histamine, and 9 ± 3 beats/min after sodium nitroprusside (NS).

Echo ejection phase indices did not change significantly after sodium nitroprusside, but fractional shortening and velocity of circumferential fibre shortening increased from $39{\cdot}4 \pm 3{\cdot}1$ per cent and $1{\cdot}30 \pm 0{\cdot}13$ s^{-1} to $55{\cdot}2 \pm 2{\cdot}1$ per cent and $1{\cdot}98 \pm 0{\cdot}14$ s^{-1} ($P < 0{\cdot}01$) during histamine, returning to $40{\cdot}2 \pm 1{\cdot}2$ per cent and $1{\cdot}36 \pm 0{\cdot}11$ s^{-1} after cimetidine.

Myocardial H_2 receptor stimulation causes a direct positive inotropic response.

Role of alpha adrenergic stimulation in arrhythmias induced by coronary reperfusion

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In chloralose anaesthetised cats alpha receptor blockade with phentolamine or prazosin or depletion of myocardial catacholamines with 6-hydroxydopamine significantly reduced the number of premature ventricular contractions (321 ± 62 *vs* 14 ± 10) induced by coronary reperfusion (35 minutes after left anterior descending occlusion) and abolished mortality caused by ventricular fibrillation which occurred in 25 per cent of controls. In contrast, beta-blockade with propranolol was ineffective. Blood flow to the ischaemic zone (assessed using radiolabelled microspheres) was not affected by phentolamine before occlusion or during the hyperaemic reperfusion phase which is temporarily associated with the onset of arrhythmias. Before coronary occlusion left stellate nerve stimulation increased idioventricular rate from 66 ± 6 to 144 ± 7 beats/min ($P < 0{\cdot}01$), a response that was blocked by propranolol, but not by phentolamine. In contrast during coronary reperfusion the increase in idioventricular rate induced by stellate stimulation was abolished by phentolamine (79 ± 10 beats/min) but not by propranolol (203 ± 14 beats/min). Infusion of an alpha receptor agonist, methoxamine (10^{-7} M) directly into the left anterior descending coronary artery via a femoral artery bypass in catacholamine depleted animals did not affect idioventricular rate before coronary occlusion but caused a significant increase (33 ± 7 to 123 ± 21 beats/min) during reperfusion. Thus, enhanced alpha receptor responsiveness appears to contribute to the electrophysiological derangements and arrhythmias associated with coronary reperfusion.

Heart rate responses after beta-blocker withdrawal

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Convincing evidence that a rebound beta-blocker withdrawal syndrome exists has been lacking despite anecdotal reports and general pharmacological expectation. We studied the heart rate responses in groups of euthyroid and hyperthyroid patients and normal volunteers, during and after treatment with propranolol or other beta-blockers. A significant rebound increase in heart rate occurred two to three days after stopping propranolol (160 mg/day for six weeks). The rebound appeared to depend on the degree of adrenergic activity, being exaggerated by standing, especially with vasodilatation or underlying hyperthyroidism, or combinations of these factors. The rebound phenomenon was also apparent in the heart rate responses to isoprenaline infusion or during the Valsalva manoeuvre. A similar rebound increase in heart rate occurred three to four days after stopping atenolol (200 mg/day), the recovery pattern suggesting an oscillation with periodicity of two days. Preliminary similar studies with oxprenolol (150 mg/day) showed a much smaller rebound. The findings confirm that

increased adrenergic responsiveness is present after withdrawal of beta-blockers.

Dissociation between myocardial glycoside uptake and inotropic response in man

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Parenteral digitalisation is employed in left ventricular failure, though improvements do not always result and inotropic responses may lag behind plasma levels, possibly because of slow uptake.

Nine patients with left ventricular failure were studied at cardiac catheterisation using the possibly rapid-acting glycoside methyl digoxin. With consent and after stabilisation the following measurements were made before and for 30 minutes after a five-minute infusion of methyl digoxin, 11·0 μg/kg: pulmonary artery end-diastolic pressure, cardiac output, thermodilution coronary sinus blood flow, coronary sinus and arterial methyl digoxin levels by validated radioimmunoassay. In six patients left ventricular pressure and dP/dt were directly measured using catheter-tip manometers. From arteriovenous methyl digoxin difference and coronary sinus flow, quantitative myocardial uptake was derived.

In all patients within 20 minutes after infusion, coronary sinus methyl digoxin levels exceeded arterial levels, indicating that myocardial glycoside uptake had passed its peak and elution had become dominant. Peak dP/dt increased linearly from mean 1800 mmHg/s^{-1} to 2250 ($P < 0·05$), but changes in cardiac output and filling pressure were variable and did not attain significance, possibly due to systemic resistance changes.

Rapid methyl digoxin uptake is confirmed, but is clearly dissociated from the inotropic response which itself is not translated into useful haemodynamic effects.

Autodecremental atrial pacing—microprocessor-based tachycardia termination system

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Five patients with recurrent tachycardias underwent electrophysiological studies with particular attention to termination of tachycardia by pacing techniques. These included programmed premature stimulation, overdrive, and underdrive pacing. Two patients had concealed accessory pathways involved in tachycardia and three patients had AV nodal reentrant tachycardia.

Autodecremental atrial pacing was employed in all patients. This system is controlled by a microprocessor interfaced with a stimulator and is activated automatically by heart rates of 160 beats/min or over. The initial stimulus is introduced at an interval less than the tachycardia cycle length, determined by a preset decrement (D). Each subsequent interval is reduced by D resulting in accelerated pacing for a preset period (P). The final pacing interval is limited to 218 ms. Both P and D are operator programmable variables.

In three patients, atrial extrastimuli or atrial overdrive initiated atrial fibrillation. Autodecremental pacing converted tachycardia to sinus rhythm in all cases.

The autodecremental mode avoids stimulation in the vulnerable period and gradual acceleration of pacing over a short period results in stimulation at different phases of tachycardia. These factors may reduce the risk of initiation of atrial fibrillation and provide a simple flexible means of tachycardia termination.

Evaluation of rate programmable permanent pacemakers using technetium-99m gated cardiac blood pool scintigraphy ($^{99}Tc^{m}$ GBPS)

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The introduction of rate programmable pacemakers has recently sharpened the problem of selecting the proper rate at which to set an implantable cardiac generator. This study examines the effect of incremental right ventricular pacing on left ventricular ejection fraction (LVEF), end-diastolic volume (EDV), and an index of the cardiac output (COI = LVEF × EDV × paced rate) measured by $^{99}Tc^{m}$ GBPS in seven patients using a variety of external rate-programmable pacemakers implanted long term.

In all patients at rates between 50 and 125 beats/min there was a progressive and significant fall in LVEF (17%, $P < 0·02$) and EDV (26%, $P < 0·005$), with a similar significant reduction of stroke volume. In contrast, COI rose significantly (27%, $P < 0·02$).

The heart rate associated with peak COI varied from patient to patient and was not related to maximal LVEF. Peak COI was generally achieved between 95 and 103 beats/min but maximum outputs were also noted at lower ventricular rates (70/min). Prediction of optimal heart rate in terms of COI was not possible.

We conclude that: (i) incremental right ventricular pacing decreases LVEF and that little benefit is conferred by ventricular paced rates greater than 100/min.

(ii) $^{99}Tc^{m}$ GBPS is a useful technique to select the optimum paced rate in individual patients with programmable generators who need maximum cardiac output with minimal battery drain.

Modern endocardial atrial and ventricular pacing leads

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Seventy-five consecutive atrial leads (January 1977 to June 1979) and 100 consecutive ventricular leads (March 1978 to June 1979) were studied. Atrial leads were 35 passive and 40 active (screw-in) fixation types: initially there was a high displacement rate from atrial appendage, but modification to the passive type and introduction of active type have eliminated displacement. Acute atrial thresholds were $0{\cdot}73 \pm 0{\cdot}15$ V at 0·5 ms. P amplitudes were $2{\cdot}31 \pm 1{\cdot}47$ mV. Eight normally functioning leads were examined chronically: thresholds $1{\cdot}24 \pm 0{\cdot}26$ V at 0·5 ms and P amplitudes $2{\cdot}36 \pm 0{\cdot}73$ mV. Three P wave sensing problems have developed since lead improvements (last 53 leads); all had P amplitude $< 2{\cdot}0$ mV acutely. Ventricular leads consisted of 30 to 10 mm tined, 50 to 2 mm tined, and 20 screw-in with three displacements in 10 mm tined lead which is now obsolete and only one exit block in the other 70 leads. Acute thresholds were $0{\cdot}44 \pm 0{\cdot}17$ V at 0·5 ms and P amplitudes $9{\cdot}01 \pm 4{\cdot}03$ mV. Four normally functioning leads were examined chronically: thresholds $1{\cdot}38 \pm 0{\cdot}55$ V at 0·5 ms, P amplitudes $5{\cdot}38 \pm 2{\cdot}63$ mV (minimum follow-up one month). It appears that atrial thresholds are higher acutely but rise less and are chronically comparable with ventricular. Thus advances in surgical technique of lead placement and in technology of lead design have completely eliminated displacement from both chambers with an incidence entrance/exit block of 2·5 per cent.

Surgery for ventricular tachycardia using operative pace mapping

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Simulation of ventricular tachycardia by pacing at operation (pace mapping) has been used in nine patients to indicate the site of origin of spontaneous ventricular tachycardia for surgical ablation.

All patients (seven male, two female, ages 36 to 65 years) had recurrent life-threatening ventricular tachycardia (unifocal in seven cases and multifocal in two cases) which was refractory to optimal conventional drug therapy, including amiodarone in seven cases.

The underlying diagnoses were coronary artery disease (six patients), valvular disease with coronary embolism (two patients) and cardiomyopathy (one patient).

The origin of the ventricular tachycardia was positively identified in all cases. Excision of the site implicated was undertaken in eight cases. Where multifocal arrhythmias were present only the focus judged clinically to be most troublesome was excised. In one patient the focus was judged to be surgically inaccessible for technical reasons.

Aneurysmectomy (five patients), aortocoronary bypass grafting (three patients), and valve replacement (two patients) were performed in addition to arrhythmia surgery.

After three to 25 months, five patients remain completely cured, requiring no antiarrhythmic drugs. One patient, who had multifocal arrhythmias, now requires only disopyramide to control the subsidiary foci. Two patients died of operative complications without recurrence of ventricular tachycardia and one, whose focus could not be excised, died suddenly three months after operation.

We conclude that: (1) the simple technique of pace mapping localises sites of ventricular tachycardia; (2) the nature of surgery for ventricular tachycardia is influenced by its site, and (3) the long-term results of surgery for ventricular tachycardia depend more on the extent and nature of the underlying ventricular disease than on the mapping and surgical techniques.

Computer system for high-speed analysis of electrocardiograms

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High-speed analysis of tape-recorded electrocardio-

grams is an important clinical and research technique. Currently available analysers perform poorly in their classification of ventricular ectopic complex pairs, ventricular tachycardia, and R on T ventricular ectopic complexes. Paper write-outs of these arrhythmias are required for corroboration but this takes more than six seconds (equivalent to six minutes on electrocardiogram) to obtain. If arrhythmias are common, and not to be missed, the tape must be stopped frequently.

Such difficulties were encountered in the analysis of electrocardiograms from 17 patients who developed primary ventricular fibrillation where 83 per cent (232/280) of all 10-second samples (with ventricular ectopic complex pairs, ventricular tachycardia, or R on T ventricular ectopic complexes) were separated by less than six minutes. A similar frequency, 86 per cent (1116/1300), was found in 21 other patients with acute myocardial infarction.

A computer analysis system has been developed to cope with these difficulties. All arrhythmias of interest are detected (both by an operator and by a Pathfinder analyser) and stored without stopping the electrocardiographic recording. Ventricular ectopic complex detector sensitivity can be increased at the expense of specificity as the electrocardiographic samples are later recalled and, if required, accepted for automatic plotting.

The computer system has improved the classification and differentiation of complex arrhythmias, reduced operator fatigue, increased accuracy, improved electrocardiograph presentation, and decreased analysis time.

Sites of origin and behaviour of arrhythmias in mitral valve prolapse: assessment using simultaneous 12 lead electrocardiography

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In 22 patients with echocardiographically confirmed mitral valve prolapse simultaneous 12 lead electrocardiograms were recorded at rest (supine) and during (a) passive head-up tilting, (b) maintained deep inspiration, and (c) graded dynamic exercise. Sites of origin of arrhythmias were determined by comparison with simultaneous 12 lead electrocardiograms obtained during electrical stimulation of various known myocardial sites in other patients during or after cardiac surgery.

Three groups of patients were identified:

(1) Eleven patients presented with chest pain or dyspnoea and had typical signs of mitral valve prolapse. Seven showed left ventricular premature beats of two distinct patterns suggesting origin in the papillary muscles. Three patients had right ventricular and two had atrial extrasystoles.

(2) Of six patients without symptoms only one had frequent right ventricular extrasystoles.

(3) Of five patients presenting with arrhythmic symptoms alone, four had frequent high right ventricular extrasystoles; one developed rapid right ventricular tachycardia on initiating exercise. One patient had second degree heart block and one showed ventricular pre-excitation.

The arrhythmias were provoked or suppressed consistently by particular manoeuvres in individual patients but showed no overall patterns of behaviour.

Thus papillary muscle arrhythmias occur commonly in symptomatic mitral valve prolapse and may have a mechanical origin, whereas right ventricular arrhythmias often occur without typical symptoms or clinical signs of mitral valve prolapse and may reflect local myocardial disease.

Echocardiographic assessment of St. Jude mitral valve prosthesis

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To assess the effects of the new bi-leaflet St. Jude prosthesis upon left ventricular filling characteristics, we determined peak rates of increase in left ventricular dimension during diastole, dD/dt, and the duration of the rapid diastolic filling period (RFP), using echocardiography in 14 patients (eight female, six male) undergoing mitral valve replacement. We compared these results with similar data obtained from 14 normal subjects and 60 patients who had had mitral valve replacement, 30 with Starr-Edwards and 30 with Björk-Shiley prostheses. All patients in this study were operated upon either for rheumatic or for degenerative mitral valve disease.

Patients	*No*	*Peak dD/dt*	*Rapid filling period*
Normals	14	16·0 ±3·2 cm/s	160 ±50 ms
St. Jude	14	13·1 ±2·6 cm/s	200 ±35 ms
Starr-Edwards	30	7·4 ±3·0 cm/s	295 ±110 ms
Björk-Shiley	30	10·5 ±4·2 cm/s	180 ±80 ms

Peak dD/dt after mitral valve replacement with the St. Jude prosthesis was significantly greater than in patients with Starr-Edwards ($P < 0·01$) or Björk-Shiley ($P < 0·02$) prostheses. The duration

of the rapid filling period in these patients was similar to those with the Björk-Shiley prostheses, but significantly less ($P < 0.01$) than in patients with Starr-Edwards prostheses. Septal motion returned to normal within two weeks in 50 per cent of patients with the St. Jude valve, whereas this was rare with Starr-Edwards and Björk-Shiley valves, only occurring with paravalvular regurgitation. In conclusion, the effects of the St. Jude valve on left ventricular filling assessed echocardiographically indicate that it approximates closely to normal and is less obstructive than either the Starr-Edwards or the Björk-Shiley prosthesis in the mitral position.

Long-term clinical and haemodynamic follow-up of patients with mitral valve repair

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One hundred and thirteen hospital survivors with mitral valve repair have been followed up from three to 19 years. Early in the series patients with commissural adhesion, light calcification, and mild mixed disease were accepted for mitral valve repair. Actuarially 82.4 ± 12.1 per cent and 65.1 ± 18.1 per cent of all patients are expected to be alive 10 and 19 years after operation, respectively. Bacterial endocarditis occurred in three (2·6%) patients. The onset of bacterial endocarditis was early in two and late in one patient. None of the patients in this series received anticoagulant treatment. Systemic embolisation occurred in six (5·3%) patients (0·61 episodes for 100 patient years). The emboli were early in three and late in the other three patients. Twenty-six (23·0%) patients underwent reoperation one to 183 months (mean 77.6 ± 11.1) after mitral annuloplasty. Early clinical deterioration (caused by dehiscence of annuloplasty in three and endocarditis in two patients) led to reoperation in five patients (19·2%) while the progression of mitral valve disease was responsible for reoperation in 21 patients (80·8%); 90.1 ± 5.9 per cent of patients are predicted to be free from reoperation five years after operation, while at 10 and 15 years the figures are 77.2 ± 13.9 per cent and 53.8 ± 23.9 per cent, respectively.

Postoperative haemodynamic investigations performed in 18 patients at a mean duration of eight-and-a-half years (range four to 11) showed significant reduction in both mean pulmonary artery and wedge pressure as compared with preoperative values. The mean diastolic gradient across the mitral valve was 10 ± 2.0 mmHg at rest and 24 ± 3.0 mmHg during exercise; 84 per cent of the patients were in grade III and IV (NYHA) preoperatively while 92 per cent of patients were in grade I and II at the latest postoperative evaluation.

Mitral valve repair provides satisfactory long-term clinical and haemodynamic improvement in a selected group of patients and should always be considered in the management of mitral regurgitation. When deterioration occurs this is usually a result of the progression of mitral valve disease.

Mitral valve replacement without long-term anticoagulants (8·5 years' experience with periocardial xenograft)

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Mitral valve replacement was undertaken in 236 patients (180 single and 56 multiple) with pericardial xenografts. There were 7·6 per cent early and 5·1 per cent late deaths and none was valve related. Between 1971 and 1976 no anticoagulants were used at any time (group 1, 67 patients). Since 1976, all patients received warfarin for the first four to six weeks after operation (group 2, 113 single, 56 multiple); 70 per cent of all patients were in chronic atrial fibrillation. One patient from group 2 and four from group 1 had mild and transient pareses at two, three, 17, 30, and 42 days after operation, respectively. None of the patients with single mitral valve replacement, from group 2, had embolic episodes. Valve thrombosis and late embolisation have not been encountered in the entire series. The incidence of embolism was 1·02 episodes per 100 patient years (0·93 for single and 1·69 for multiple). Actuarially 97.1 ± 1.4 per cent of all patients are expected to be free from embolism at one and nine years after operation. The ratio of early embolism between group 1 and group 2 was 10:1.

The published data for mitral replacement with porcine xenografts show a higher embolic rate (3·8 to 5·3 episodes per 100 patient years) with a significant number of patients on long-term anticoagulants.

The lack of valve thrombosis and the very low embolic rate in mitral valve replacement with pericardial xenografts without long-term anticoagulants is a result of the superior hydraulic characteristics of this valve. Short-term postoperative anticoagulation is beneficial for patients with mitral and multiple valve replacement.

Cross-sectional echocardiographic recognition of vegetative endocarditis

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The cross-sectional echocardiographic (CSE) findings in 19 patients thought diagnostic of vegetations of endocarditis were reviewed in order to characterise their appearance and to determine their specificity. Of the 19 patients with cross-sectional echocardiographic features of vegetations, 13 had proven endocarditis diagnosed on strong clinical evidence in five (three aortic, one mitral, one both valves) with surgical confirmation in the remaining eight (all aortic). Six patients were incorrectly diagnosed as having vegetations (one aortic homograft, five mitral). The cross-sectional echardiographic appearance of aortic valve vegetations (12 patients) were of dense homogeneous echo masses in 10 patients, with evidence of diastolic prolapse into left ventricular outflow tract in six. All patients with prolapsing vegetations underwent early aortic valve replacement. Two patients had non-homogeneous atrioventricular echo masses which prolapsed and at operation had cusp rupture with healed vegetations. Mitral valve vegetations observed in two patients were dense and homogeneous and prolapsed through the mitral valve in diastole in both cases. Of the six patients incorrectly diagnosed as having vegetations, the appearances were less characteristic, being less dense and less homogeneous in all six cases. One patient had a leaking atrioventricular homograft with cusp degeneration and prolapse at surgery. Of the remaining five patients, two had previously treated endocarditis and presumably healed vegetations, two patients had angiographic mitral valve prolapse only, and one patient had suspected rupture of mitral valve chordae.

Cross-sectional echocardiography is valuable in confirming the presence of vegetations when they are dense homogeneous and prolapse into the left ventricular outflow tract from the atrioventricular position. Using less characteristic criteria, particularly when defining mitral valve vegetations, may lead to errors in diagnosis.

Estimation of cardiac output by echocardiography using left atrial dimensions

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Cardiac output has been estimated at echocardiography using two assumptions:

(1) The rate of increase of left atrial volume during ventricular systole is representative of pulmonary venous flow throughout the cardiac cycle.

(2) Left atrial volume (LAV) is given by: $LAV = 7{\cdot}0/(2{\cdot}4+D) \times D^3$, where D = LA dimension (in cm) measured from the posterior aortic wall to the LA posterior wall.

Cardiac output was then calculated as:

$= (([LAV]\text{late ventricular systole} - [LAV]\text{early ventricular systole}) \times 60)/\text{Time interval between LA dimension measurements (s)}$

Ten cardiac cycles were analysed and the means taken.

The validity of the technique was assessed by comparison with dye dilution measurement of cardiac output in 22 consecutive patients after cardiac catheterisation. Thirteen had valvular disease, seven had coronary heart disease, one had cardiomyopathy, and one was normal. Cases with mitral reflux or shunts were excluded.

Mean cardiac output by dye dilution was 3·92 (SD 1·24) l/min (range 1·8 to 6·6): by echocardiography the mean was 3·80 (SD 1·11) l/min. The correlation coefficient was 0·92.

The left atrial echocardiographic dimension used is more easily obtained than simultaneous echoes of the left ventricular walls, and, unlike cardiac output estimation from left ventricular volumes, the calculation appears not to be affected by left ventricular dysynergy or aortic reflux.

Cross-sectional and M-mode echocardiography in acute aortic dissection

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Eighteen patients who suffered thoracic aortic dissections were studied using cross-sectional and M-mode echocardiography. The echocardiogram was recorded and interpreted before angiography.

Recordings were considered highly suggestive of dissection involving the ascending aorta if they showed a dilated and abnormal aortic root together with evidence of either pericardial effusion or aortic regurgitation.

Fifteen patients suffered DeBakey type I or type II dissections. The echocardiogram was positive by the above criteria in 13 of these. Cross-sectional echocardiography enhanced the imaging

of aortic root anatomy and also allowed visualisation of an intimal flap. In addition, a new diagnostic feature was observed on M-mode recordings in five patients. This consists of a bizarre low-frequency movement of the aortic cusps during systole. Left ventriculography suggests that this results from interruption of the cusp attachments of the valve.

In the remaining three patients, a DeBakey type III dissection was present. Only one of these was detected. Cross-sectional echocardiography performed from the suprasternal notch was used in this case.

This study suggests that the sensitivity of echocardiography in detecting aortic dissection may be higher than previously described. The inclusion of a new echocardiographic criterion enhances the sensitivity of the M-mode technique.

Aortocoronary bypass surgery for prophylaxis only: what is the size of the problem?

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Retrospective and prospective data suggest that certain patients with coronary artery disease benefit prophylactically from surgery. An oversimplified definition of the extent of the disease has been used. We have defined disease strictly, selecting patients with coronary indices $\leqslant 25$ per cent including all with major proximal stenoses of the three main coronary trunks and left main stenosis. Of 416 such patients, 158 were treated medically and 258 surgically. The difference in survival at one year is 19 per cent, at two years 33 per cent, and at three years 43 per cent, and is similar for normal or abnormal ventricles. It is therefore important to select these patients for operation. They are drawn from over 3000 patients with known or suspected coronary disease who underwent coronary arteriography; 77 per cent had grade 2 angina or worse and only 9 per cent were asymptomatic, 88 per cent had a maximum exercise performance of $\leqslant 600$ kpm including 87 per cent of the asymptomatic or grade 1 angina patients.

These data suggest that the majority of patients likely to benefit prophylactically from surgery will also have symptoms that warrant its consideration; most of the small number of patients who do not could be selected by exercise testing. The often-stated problem that investigation centres would be unable to cope with the large numbers of coronary arteriograms necessary to select the relatively small number of patients may not, in fact, exist.

Coronary surgery after recurrent myocardial infarction: progress of trial comparing surgical with conservative management for asymptomatic patients with advanced coronary disease

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A randomised trial of surgical versus conservative treatment has been carried out for male patients up to 60 years of age who have recovered from a recurrent myocardial infarct. Of 205 cases considered, 100 were relatively asymptomatic and had coronary anatomy favourable for bypass grafting; these patients fulfilled the trial conditions and were randomised (50 surgical, 50 medical). In 40 cases (elective medical group) randomisation for surgery was not considered justifiable because of relatively unfavourable coronary anatomy and/or severe left ventricular dysfunction. Twenty patients had elective surgery because of disabling angina despite full medical treatment or for significant left main coronary stenosis. In 45 cases investigation of coronary anatomy was not undertaken because of medical contraindication or patient reluctance to enter the study.

Actuarial survival curves (mean follow-up 3·5 years) show an annual mortality rate of 3 to 4 per cent per year for all investigated patients, and no advantage for any treatment group. Results suggest that in the absence of disabling angina or left main coronary stenosis, coronary artery surgery need not be advised for survivors of recurrent infarction who have severe coronary artery disease. Moreover, the prognosis for this group of patients with conservative treatment appears to be better than has been previously described.

Anomalous origin of left coronary artery from anterior aortic sinus: potential cause of sudden death—anatomical characterisation and surgical treatment

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Three patients with the syndrome of anomalous origin of the left coronary artery from the anterior aortic sinus presented at the ages of 14, 30, and 33 years, respectively, with angina of effort. There were associated electrocardiographic changes in

two. One patient had additional aortic regurgitation. Coronary angiography showed characteristic appearances of the anomaly. Two patients were treated surgically. The left coronary artery arose from the anterior sinus in very close relation to the intercoronary commissure and passed deep to that commissure in the substance of the aortic wall for a distance of approximately 1 cm before following its normal course. Correction of the anomaly was achieved in one patient by temporary detachment of the commissure, and deroofing the left coronary artery to the mid-point of the left coronary sinus and by homograft replacement of the aortic valve and root with anastomosis of the left coronary artery to the coronary orifice of the homograft in the second. Both patients are symptom-free, with no evidence of perioperative or late myocardial infarction 12 and 24 months after operation. Repeat exercise test and coronary angiography in the younger patient have shown normal findings.

Late results of resection of left ventricular aneurysm

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Between August 1969 and July 1979, 111 patients underwent resection of left ventricular aneurysm. Of these, 49 were operated on before July 1974, thus giving a minimum follow-up period of five years. There were 42 men and seven women, ranging in age between 28 and 65 years (mean 52·6). The presenting symptom was angina in 10 (20%), dyspnoea in 23 (46%), and both in 12 (24%). Arrhythmia was present in six. Pre-existing systemic hypertension was present in four. Additional coronary grafting was performed in 27 patients (55%). There were four (8%) early and 15 (31%) late deaths (mean follow-up 59·8 months). Actuarial survival was 81·8 per cent at three years, 69·6 per cent at five years, and 61·3 per cent at seven years. The cause of late death was cardiac in 12 (80%), other conditions in two (13%), and unknown in one (7%). Late survival was 90 per cent for patients presenting with angina, 70 per cent for those with shortness of breath, and 25 per cent for those presenting with both. There were no early or late deaths in patients operated on for arrhythmia. Of the surviving patients 83 per cent were symptom-free or improved, and 17 per cent the same or worse.

It is concluded that the late results of resection of left ventricular aneurysm are probably better than the natural history of this condition and are related to the presenting symptoms.

Incidence and significance of late myocardial infarction after coronary artery bypass grafting

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Between October 1969 and May 1979, 978 patients underwent coronary artery bypass grafting (CABG) at Harefield Hospital. Of these, 783 had isolated CABG and 2112 grafts were inserted (2·6 grafts per patient). Late myocardial infarction, as diagnosed by clinical, enzymatic, or electrocardiographic criteria, was diagnosed in 34 (4·3%) of the surviving patients (mean follow-up of 40·5 months). There were 20 anterior and 14 inferior infarcts. Infarction occurred in the territory of grafted vessels in 28 (82%), while in six patients (18%) it was in the area of a non-grafted vessel. There were eight deaths (22%) of which five occurred within four weeks of infarction. Twenty-six patients survived infarction and were followed up for a period of two to 70 months (mean 26·6); 18 (69%) had recurrence of symptoms. Twenty-one patients were reinvestigated by repeat angiography. The patency rate of grafts to infarcted areas was 47 per cent. Left ventricular segmental wall motion showed deterioration in 14 out of 18 (78%) and was unchanged in four (28%).

It is concluded that late myocardial infarction after CABG is uncommon, carries a bad prognosis in terms of symptomatic outcome and left ventricular segmental wall motion analysis, and is related to progression of disease in approximately 50 per cent.

Atrial and ventricular human myosin during development

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Myosin, the major contractile protein of the myofibril was prepared from human myocardium excised soon after death. The low molecular weight light chain subunits involved in actin-binding and actomyosin ATP-ase activity were examined using two-dimensional polyacrylamide gel electrophoresis. The rate of ATP-ase activity determines the contractile speed of the muscle, and abnormal activities have been observed in various pathological conditions, for example hypertrophy and cardiac failure. Myosin from the free right and left adult ventricle

wall was identical with two light chains, VLC-1 and VLC-2 of molecular weight 25 900 and 18 000, respectively. Adult right and left atrial myosin light chains (ALC-1 and ALC-2) were different electrophoretically from adult VLC-1 and VLC-2. Adult atrial myosin possessed a Ca^{2+} ATP-ase activity double that of ventricular myosin. Myosin from the right and left ventricle of 18 to 21-week human fetuses contained not only adult type VLC-1 and VLC-2 but significant amounts of ALC-1. Similar amounts of ALC-1 were present at 34 weeks but only small amounts were present eight months after birth. The presence of different myosins with varying activities in the normal myocardium during development may be the basis of similar changes in activity observed in the hypertrophied heart.

Morphology and fibre orientation of human left ventricle

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Models of cardiac function which assume the left ventricle to be homogeneous and isotropic have been proposed on the basis of both pressure and volume measurements and experiments on isolated cardiac muscle. It would be more appropriate to base such models on knowledge of the morphology of the human left ventricle. To provide this information we studied the anatomy and fibre orientation in 10 human hearts from patients without clinical evidence of cardiovascular disease. Left ventricular fibres can be traced from the mitral annulus, aortic and pulmonary roots. They pass either longitudinally to the apex and thence deeply to enter the septum or more obliquely around the left ventricular free wall to enter the right ventricle. Reflection of the outer fibres reveals a middle layer arranged more perpendicularly to the long axis. Deep to this is a third layer at about 90 degrees to the outermost layer. These divisions into layers illustrate the general disposition of fibres. There are important regional variations. The septum is largely composed of fibres from the middle layer of the left ventricle. Around the mitral orifice the arrangement is circular while it is longitudinal at the apex. Fibre orientation in man thus differs significantly from that previously described in other species, and is likely to be a major determinant of normal and abnormal left ventricular wall mechanics.

Immunofluorescent studies of sera and cardiac muscle from patients with cardiomyopathy

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Although there is controversy concerning the presence of circulating heart antibodies in patients with primary cardiomyopathies, the present consensus is that they are not present to any significant extent. However, Das *et al.*[1] have suggested that this lack of circulating antibody may be a result of its binding to cardiac muscle.

We have simultaneously obtained sera and left or right ventricular biopsies from 31 patients undergoing diagnostic cardiac catheterisation. Eight of these had primary cardiomyopathy, five hypertrophic cardiomyopathy, three alcoholic cardiomyopathy, and three myocarditis. The remainder were found to have other significant cardiac lesions.

Cardiac muscle and sera were examined by two independent observers unaware of the final clinical diagnosis. Using the direct immunofluorescent technique, biopsies were examined for the presence of bound IgG, IgA, IgM, fibrin, and complement. An indirect immunofluorescent method using baboon heart and the patient's own biopsy as substrates was used to detect circulating heart antibodies.

There was no correlation between the presence or absence of weakly reacting serum antibodies and the variations in intensity of direct fluorescence seen between biopsies.

We conclude that there is no evidence that the lack of circulating heart antibodies in patients with cardiomyopathy is a result of the binding of these antibodies to cardiac muscle. These results are further evidence against a primary role of humoral antibodies in the pathogenesis of primary cardiomyopathies.

Reference

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Diagnostic and prognostic aspects of endomyocardial biopsies

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Morphological evaluation of endomyocardial biopsies has been undertaken by semiquantitative and quantitative measurements in 361 biopsies from 201 patients suspected to suffer from cardiomyopathy. The findings have been related to clinical and haemodynamic indices such as length of history, ejection fraction, and left ventricular end-diastolic pressure.

At light microscopical level pooling of quantitative results shows characteristic features of some entities, and grouping of patients is marginally more accurate than that achieved by conventional histological examination. Normal tissue can be separated from pathological samples, but sufficient basis for a definite distinction between individual patients with abnormal histology has not been established. Furthermore, no correlation between quantitative morphology and clinical or haemodynamic information has been found.

A group of 48 patients has been analysed semiquantitatively at electron microscopical level. No relation with haemodynamic measurements or assessment of prognosis can be established.

Analysing endomyocardial biopsies from hearts at necropsy it has been found that five biopsies at least are required to reflect accurately the state of the myocardium.

It is concluded that endomyocardial biopsy plays an important role as an adjunct to diagnosis, that morphometry increases accuracy of the findings, but that prognostic evaluation should be interpreted with the greatest caution.

Abnormal electrocardiograms in athletes

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Nine athletes aged between 18 and 41 had electrocardiograms recorded during routine examinations. The electrocardiograms were regarded as abnormal, six on account of left ventricular hypertrophy with T wave changes, one because of anterior Q waves, and two because of T wave changes only. All the athletes had been advised to relinquish their sporting activities and ambitions but had been referred for a further opinion.

The referral diagnoses were cardiomyopathy (in three), hypertrophic cardiomyopathy (in two), ischaemic heart disease (in two), and uncertain (in two).

Each patient had a full examination, rest and exercise electrocardiogram, chest *x*-ray, echocardiogram, and cardiac catheterisation. Coronary arteriograms were performed in seven.

No patient was hypertensive. Soft third sounds, fourth sounds (palpable in three), and systolic murmurs were common. The chest *x*-ray was normal in all. The echocardiogram was normal in five, showed symmetrical hypertrophy in two, and asymmetrical septal thickening in two. The left ventricular angiograms were thought to show hypertrophy in three, but only one was definitely abnormal. This showed discrete anterior wall dyskinesia: four of the exercise tests resulted in normalisation of the T wave changes. All coronary arteries appeared widely patent (including the arteries of the patient with the abnormal angiogram). Eight of these patients were considered to have normal hearts and were advised to resume their training. One was thought to have suffered a traumatic infarct. Two patients had insurance loading withdrawn and one regained his civil pilot's licence.

Harm may be done if the probable normality of 'abnormal' electrocardiographic abnormalities in athletes is not recognised. Though normalisation of T wave changes on exercise and a normal echocardiogram help to exclude significant pathology, invasive studies are fully justified when the possibility of heart disease threatens to disrupt the patient's life.